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Chapter 5

Idiopathic Ventricular Arrhythmias

Takumi Yamada

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Abstract

Idiopathic ventricular arrhythmias (VAs) occur with a mechanism that is unrelated to myocardial scar. Idiopathic VAs most commonly occur in patients without structural heart disease, but can occur in those with structural heart disease. Idiopathic VAs present as a sustained or a non-sustained ventricular tachycardia or premature ventricular contractions. Imaging examinations such as echocardiography, nuclear tests, and cardiac magnetic resonance imaging are helpful for excluding any association of an idiopathic VA occurrence with myocardial scar. For the past two decades, the sites of idiopathic VA origins, commonly endocardial but sometimes epicardial, have been increasingly recognized. Idiopathic VAs usually originate from specific anatomical structures and exhibit characteristic electrocardiograms based on their anatomical background. Idiopathic VAs are basically benign, but they require medical treatment or catheter ablation when idiopathic VAs are symptomatic, frequent, or cause tachycardia-induced cardiomyopathy. This book chapter describes the up-to-date information on the prevalence of idiopathic VA origins relevant to the anatomy, diagnosis, and treatment of idiopathic VAs.

Keywords: idiopathic, ventricular tachycardia, anatomy, diagnosis, treatment

1. Introduction

Idiopathic ventricular arrhythmias (IVAs) present as ventricular tachycardias (VTs) or premature ventricular contractions (PVCs) whose mechanisms are not associated with a myocardial scar. IVAs commonly occur in patients without structural heart disease (SHD), but can occur in those with SHD [1–3]. Classically, VTs originating from the right ventricular outflow tract (RVOT) and the left posterior fascicle are well known as IVAs. However, for the past two decades, IVAs originating from other endocardial and also epicardial sites have been increasingly recognized (**Figure 1**) [3]. IVAs usually originate from the specific anatomical

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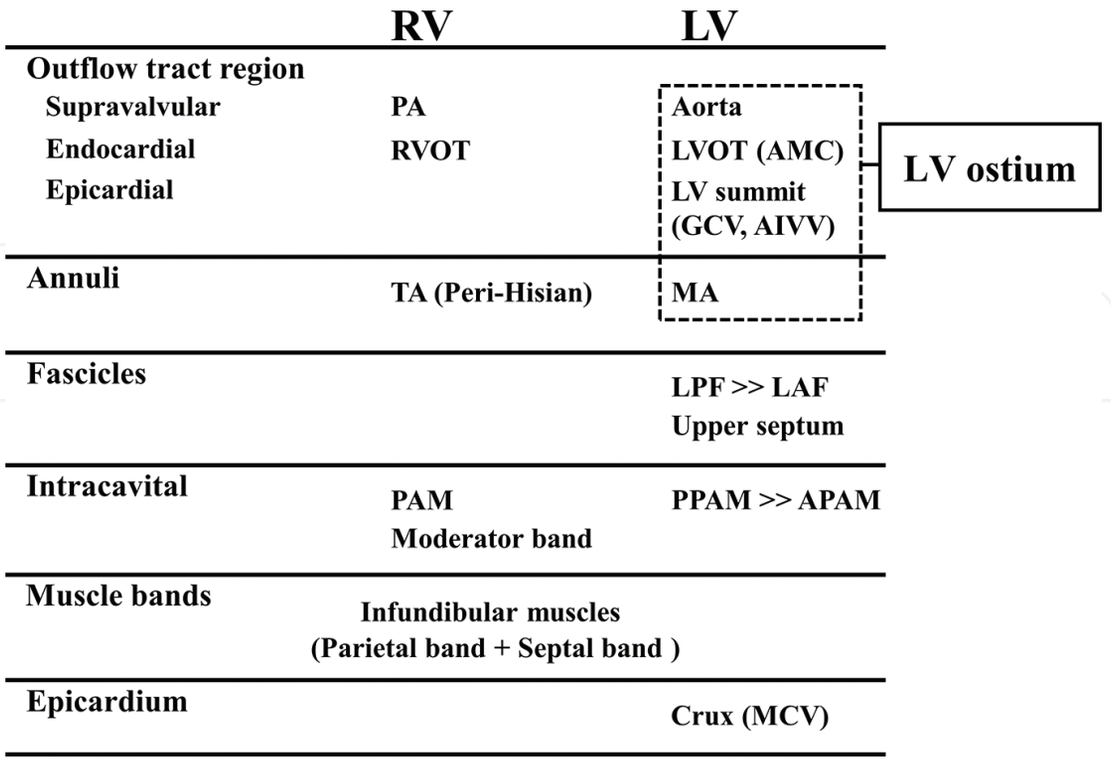


Figure 1. Idiopathic ventricular arrhythmia origins. AIVV, anterior interventricular vein; AMC, aorto-mitral continuity; APAM, anterolateral papillary muscle; GCV, great cardiac vein; LAF, left anterior fascicle; LPF, left posterior fascicle; LV, left ventricle; LVOT, LV outflow tract; MA, mitral annulus; MCV, mid-cardiac vein; PA, pulmonary artery; PAM, papillary muscle; PPAM, posteromedial papillary muscle; RV, right ventricle; RVOT, RV outflow tract; TA, tricuspid annulus.

structures and exhibit characteristic electrocardiograms based on their anatomical background. Basically, IVAs are benign and not life-threatening, but are often symptomatic and also can cause tachycardia-induced cardiomyopathy [4, 5]. Therefore, it is important for cardiologists to update their knowledge about IVAs. This chapter describes the current expertise on the prevalence of IVA origins relevant to the anatomy, diagnosis, and treatment of IVAs.

2. Prevalence of IVA origins relevant to the anatomy

The sites of IVA origins have been identified by electrophysiological mapping and confirmed by successful catheter ablation. The most common site of IVA origins is the ventricular outflow tract [1, 6]. IVAs originate more often from the RVOT than from the left ventricular outflow tract (LVOT). In the RVOT, the septum is a more common site of IVA origins than the free wall. The most common site of IVA origins in the LVOT is the aortic root followed by the sites underneath the aortic sinus cusps (ASCs) (**Figure 2**) [7, 8]. Especially, the site underneath the left coronary cusp (LCC) is located in front of the mitral annulus (MA) and is termed the aorto-mitral continuity (AMC). The MA is also one of the major sites of IVA origins [9, 10]. The anteromedial aspect of the MA may overlap with the AMC. Anatomically, the aortic and mitral valves are in a direct apposition and attach to the elliptical opening at the base of the left ventricle (LV)

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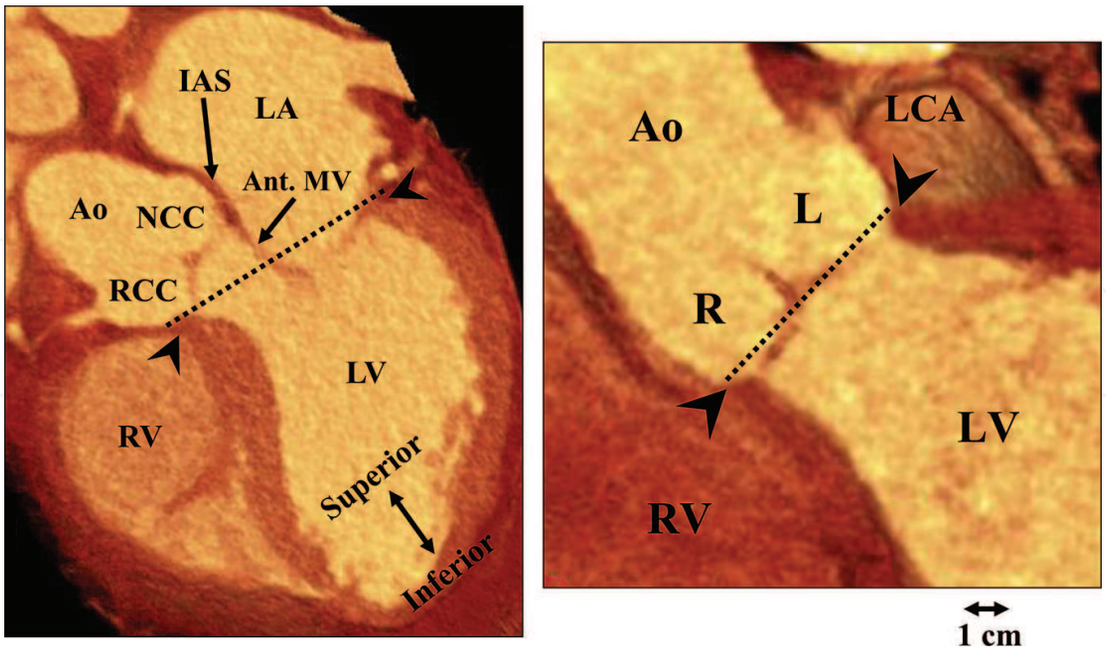


Figure 2. Two-dimensional computed tomography (CT) images showing the relationships between the ventricular myocardium and aortic sinus cusps. The arrowheads and dotted line in the left panel indicate the ostium of the left ventricle. The arrowheads in the right panel indicate the superior edge of the ventricular myocardium connecting with the left coronary cusp and right coronary cusp (RCC), and the dotted line the ventriculo-arterial junction. Ant, anterior; Ao, aorta; IAS, interatrial septum; L, left coronary cusp; LA, left atrium; LCA, left coronary artery; LV, left ventricle; MV, mitral valve; NCC, noncoronary cusp; R, right coronary cusp; RV, right ventricle. This figure was cited from Ref. [7] with permission.

known as the LV ostium [11, 12] (**Figure 2**). Because there is no myocardium between the aortic and the mitral valves (fibrous trigone), most idiopathic LV ventricular arrhythmias (VAs) can originate from along the LV ostium. The LV myocardium comes in direct contact with the aorta at the base of the ASCs (**Figure 2**). When IVAs arise from the most superior portion of the LV ostium (the aortic sinus of valsalva), they can be ablated within the base of the ASCs. It has been reported that some IVAs can be ablated from the junction (commissure) between the left and the right coronary cusps (L-RCC) [13]. In these VAs, catheter ablation from underneath the ASCs is often required for their elimination. Anatomically, the superior end of the LV myocardium makes a semicircular attachment to the aortic root at the bottom of the right and left coronary cusps. However, because of the semilunar nature of the attachments of the aortic valvular cusps, the superior end of the LV myocardium is located underneath the aortic valves at the L-RCC (**Figure 2**). Therefore, IVAs that can be ablated at the L-RCC should be classified into the same group as the VAs that can be ablated within the ASCs. In this setting, these IVAs may be defined as IVAs arising from the aortic root [7]. It has been reported that IVAs can rarely be ablated from within the noncoronary cusp of the aorta (NCC) [7, 14, 15]. Spatially, the aortic root occupies a central location within the heart, with the NCC anterior and superior to the paraseptal region of the left and right atria close to the superior atrioventricular junctions (**Figure 3**) [12]. In normal human hearts, the NCC is adjacent to the atrial myocardium on the epicardial aspect of the interatrial septum. When atrial tachycardias arise from that region of the atria, those atrial tachycardias can be ablated from within the NCC. It is considered that the NCC does not directly come in contact with the ventricular myocardium

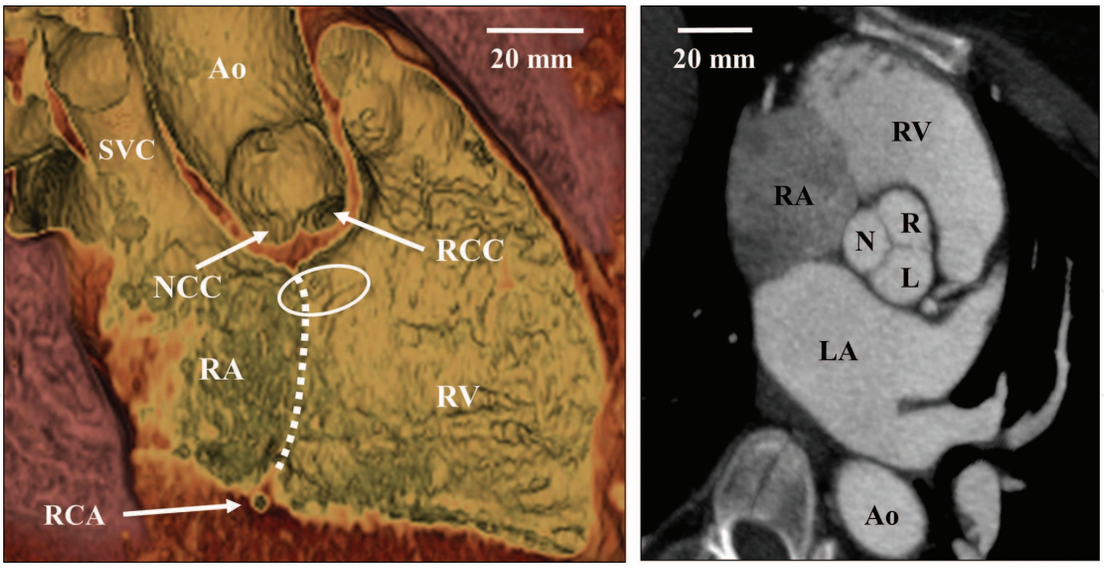


Figure 3. Two-dimensional (right panel) and three-dimensional (left panel) CT images. The dotted line indicates the tricuspid annulus and solid circle the right ventricular His bundle (HB) region. L, left coronary cusp; N, noncoronary cusp; RA, right atrium; RCA, right coronary artery; SVC, superior vena cava. The other abbreviations are as in the previous figures. This figure was cited from Ref. [15] with permission.

(**Figure 3**). However, it has been reported that some IVAs can be ablated from within the NCC [14]. The clinical observation that a noncoronary sinus of valsalva aneurysm can rupture into the right ventricle (RV), as well as the right atrium, supports the assumption that the NCC may be attached to the ventricular myocardium where IVAs can arise from. Some IVAs can be ablated from within the pulmonary sinus cusps [16]. The ventricular muscle is attached to the pulmonary sinus cusps (PSCs) in the RVOT like the ASCs in the LVOT. The ventricular muscle extends above the PSCs, but it should be noted that ventricular myocardial extensions never occurs in the aorta [12]. The ventricular muscle may appear to extend above the right coronary cusp (RCC) because of the specific nature of the interventricular septum. However, the superior end of the *left* ventricular muscle attaches to the RCC, and the *right* ventricular muscle attaching to the *left* ventricular muscle underneath the RCC runs in the interventricular septum up to the PSCs, which is located above the ASCs. In fact, the *right* ventricular muscle in the interventricular septum is separated from the RCC and aorta by a loose connective tissue. When IVAs arise from the ventricular muscle underneath the ASCs and PSCs or above the PSCs, catheter ablation within the ASCs and PSCs is required to cure those VAs because those VA origins are likely to be epicardial.

IVAs can originate from the atrioventricular annuli including the MA [9, 10] and tricuspid annulus (TA) [17]. IVAs originating from the MA and TA account for 5 and 8% of all IVAs, respectively. MA VAs can originate from any of the regions along the MA except the septal aspect where the fibrous trigone is located with no ventricular myocardium, but the anterolateral and posteroseptal aspects of the MA are the most common and second most common sites of MA VA origins, respectively [9, 10]. TA VAs can originate from any regions along the TA, but more often originate from the septal aspect, especially in the anteroseptal or para-Hisian region than the free wall [17].

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IVAs can arise from the intracavital structures including the papillary muscles (PAMs) [18–22] and moderator band (MB) [23]. PAM VAs account for approximately 7% of patients with IVAs [18–22]. LV PAM VAs are known to arise more commonly from the posteromedial PAM than from the anterolateral PAM [20]. The sites of the PAM VA origins are limited to the base of the PAMs. IVAs can rarely originate from the PMs in the RV [22]. IVAs can arise from all three RV PAMs, but half of them arise from the septal PAM [22]. It has been recently reported that the MB rarely can be a source of IVAs including PVCs, VTs, and ventricular fibrillation [23]. Anatomically, the MB is considered to be a part of the septomarginal trabeculation, crossing from the septum to the RV free wall and supporting the anterior PAM of the tricuspid valve (**Figure 4**) [23].

Most recently, it has been reported that IVAs can arise from the muscular bands in the RV [24, 25]. The RVOT is the most common site of IVA origins. In the RV, the TA is the second most common site of IVAs, and less commonly idiopathic VAs can originate from some RV muscles [3, 17, 22, 23]. Anatomically, the muscles of the RV may be divided into three groups: (1) trabeculae, (2) papillary muscles of the tricuspid valve, and (3) infundibular muscles (**Figure 5**). The muscles of the infundibulum are thick muscular bands, consisting of the septal and parietal bands. The junction of these two bands is often indicated by a raphe or a ridge extending from the superior papillary muscle to the nadir of the posterior pulmonary leaflet. This junction has been

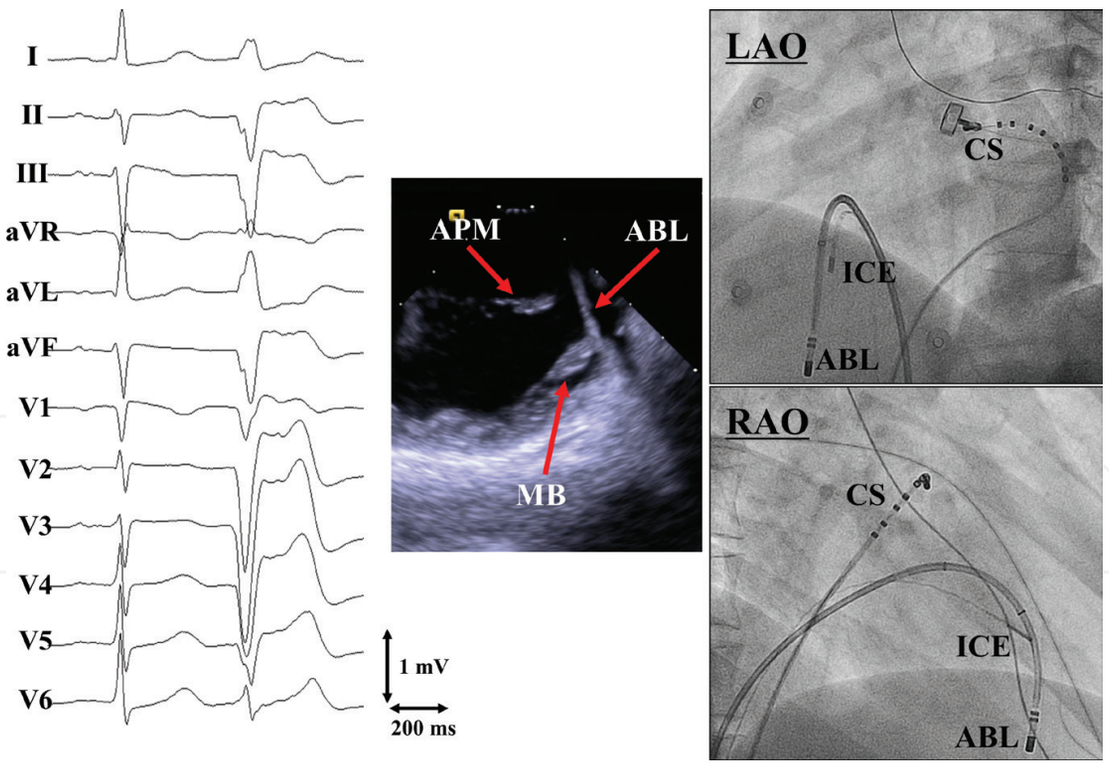


Figure 4. Twelve-lead electrocardiograms exhibiting a premature ventricular contraction originating from the moderator band (MB) (left panel) and an intracardiac echocardiographic image (middle panel) and fluoroscopic images (right panels) exhibiting the successful ablation site of the premature ventricular contraction originating from the MB. ABL, ablation catheter; APM, anterolateral papillary muscle; CS, coronary sinus; ICE, intracardiac echocardiography catheter; LAO, left anterior oblique; RAO, right anterior oblique. The other abbreviations are as in the previous figures. This figure was modified from Ref. [10] with permission.

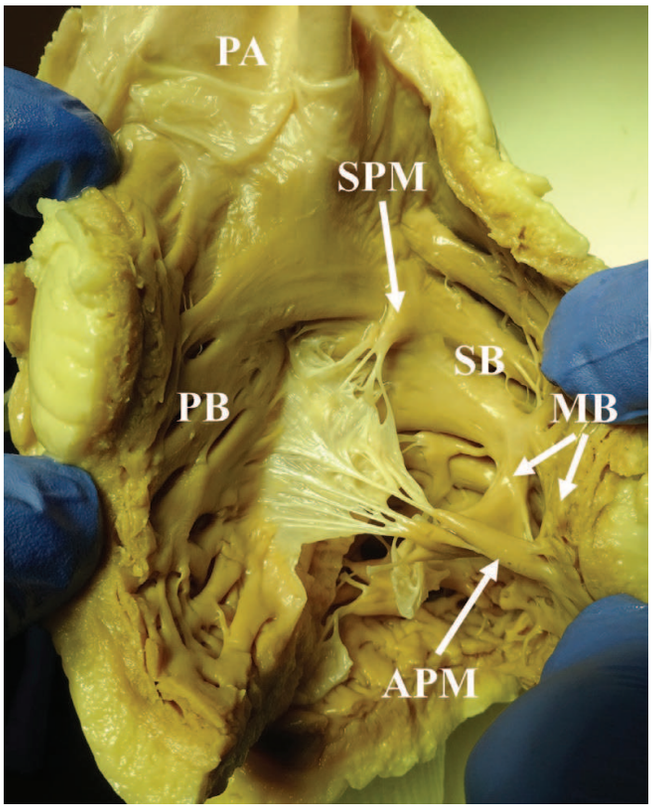


Figure 5. Autopsy heart exhibiting the infundibular muscles. PA, pulmonary artery; PB, parietal band; SB, septal band; SPM, superior papillary muscle. The other abbreviations are as in the previous figures. This figure was adapted from Ref. [25] with permission.

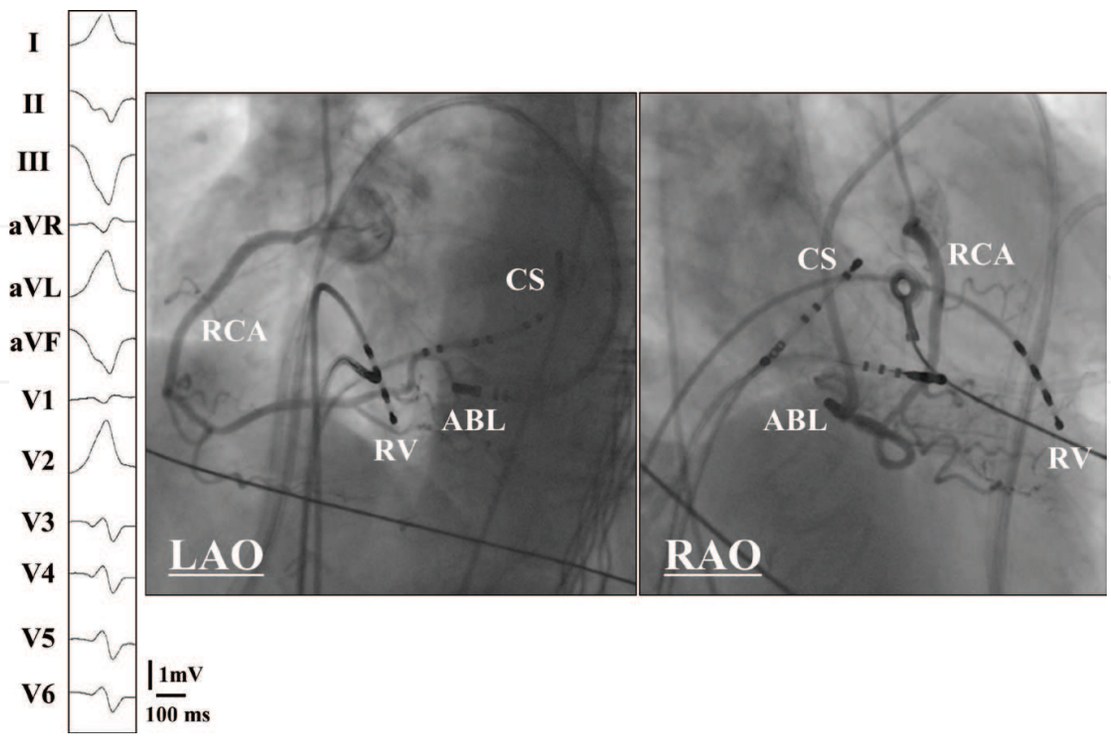


Figure 6. Twelve-lead electrocardiograms exhibiting the ventricular arrhythmia originating from the crux of the heart (left panel) and fluoroscopic images exhibiting its successful ablation site. The abbreviations are as in the previous figures. This figure was adapted from Ref. [30] with permission.

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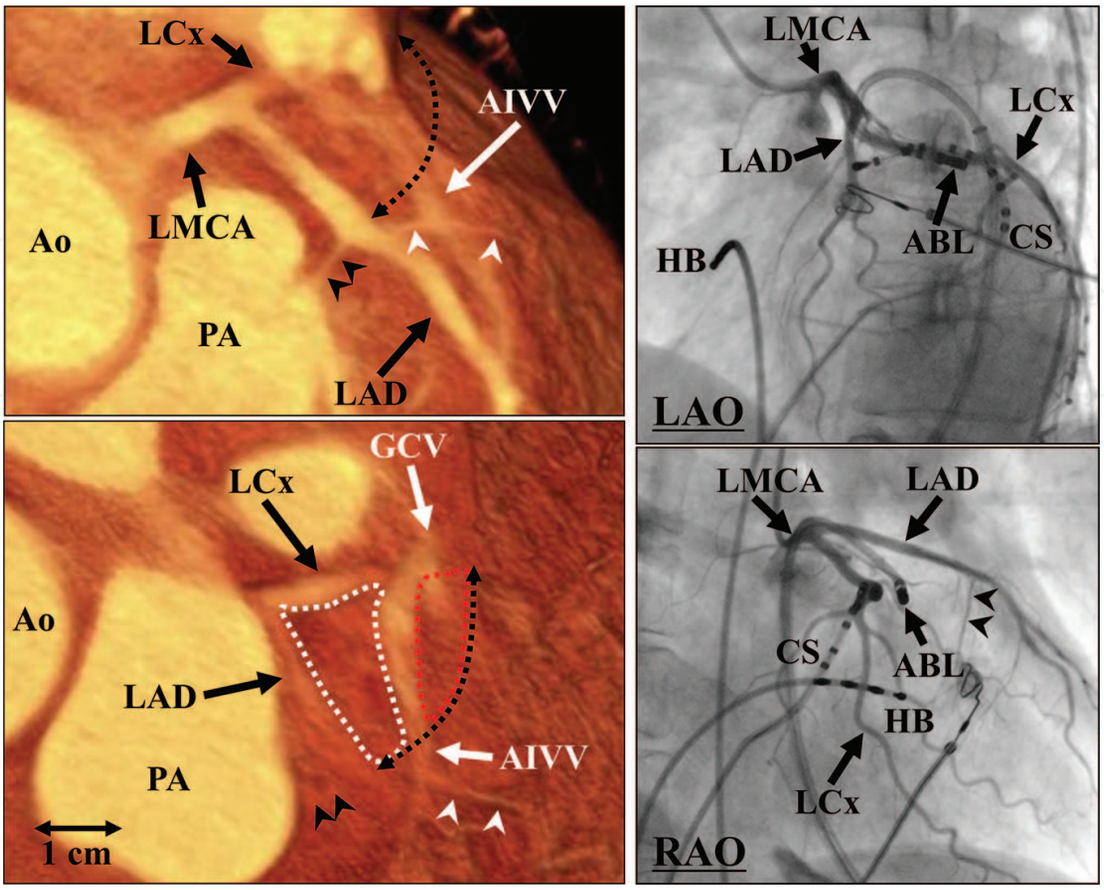


Figure 7. CT (left panels) and fluoroscopic (right panels) images exhibiting the LV summit. The LV summit was defined based on the fluoroscopy and coronary angiography as the region on the epicardial surface of the LV near the bifurcation of the left main coronary artery that is bounded by an arc (black dotted line) from the left anterior descending coronary artery (LAD) superior to the first septal perforating branch (black arrowheads) and anteriorly to the left circumflex coronary artery (LCx) laterally. The great cardiac vein (GCV) bisects the LV summit into a superior portion surrounded by the white dotted line (the *inaccessible area*) and an inferior portion surrounded by a red dotted line (the *accessible area*). The white arrowheads indicate the first diagonal branch of the LAD. AIVV, anterior interventricular cardiac vein; HB, His bundle; LMCA, left main coronary artery; PA, pulmonary artery. The other abbreviations are as in the previous figures. This figure was cited from Ref. [31] with permission.

termed the crista supraventricularis. An extension of the septal band is the MB, which usually extends inferiorly to the site of attachment of the anterior papillary muscle in the anterior wall. The parietal band extends across the tricuspid orifice onto the anterior wall, fading out above the area of the attachment of the anterior papillary muscle. IVAs rarely arise from the infundibular muscles, and parietal band IVAs are approximately three times as prevalent as septal band IVAs.

IVAs can arise from the Purkinje network, most commonly from the left posterior fascicle followed by the anterior and septal fascicles [21, 26, 27]. These IVAs most often present as reentrant VTs, but sometimes as VTs or PVCs with a focal mechanism. The left anterior fascicle runs along the MA. The left septal fascicle is located between the left anterior and posterior fascicles, and there is a normal variation in its origin and distribution. The peripheral Purkinje network extends to the surface of the PAMs and MB. Therefore, these VAs have to be differentiated from IVAs originating from the PAMs, MB, and atrioventricular annuli.

IVAs arise commonly from the endocardial side, but can arise from the epicardial side [27] and rarely from the intramural site [28, 29]. There are two major sites of origin of idiopathic

epicardial VAs such as the crux of the heart [30] and LV summit [31]. Anatomically, the crux of the heart is formed by the junction of the atrioventricular groove and the posterior interventricular groove and corresponds roughly to the junction of the middle cardiac vein and coronary sinus, near the origin of the posterior descending coronary artery (**Figure 6**) [30]. A region of the LV epicardial surface that occupies the most superior portion of the LV has been termed the LV summit by McAlpine (**Figure 7**) [11, 31]. The LV summit is bounded by the left anterior descending coronary artery and the left circumflex coronary artery. This region near where the great cardiac vein (GCV) ends and the anterior interventricular cardiac vein begins is one of the major sources of epicardial IVAs. The LV summit is bisected by the GCV into an area lateral to this structure that is accessible to epicardial catheter ablation (the *accessible area*) and a superior region that is inaccessible to catheter ablation due to the close proximity of the coronary arteries and a thick layer of epicardial fat that overlies the proximal portion of these vessels (the *inaccessible area*) [31]. The prevalence of LV summit VAs has been reported to account for 12% of idiopathic LV VAs. Among these VA origins, 70, 15, and 15% of them have been identified within the GCV, accessible area, and inaccessible area, respectively.

3. Diagnosis of IVAs

3.1. Imaging

IVAs are defined as VAs originating from normal ventricular myocardium. Therefore, any association of myocardial scar with an occurrence of VAs has to be excluded for a diagnosis of IVAs. Echocardiography and exercise stress testing are basic examinations to demonstrate no evidence of SHD. However, IVAs can occur in patients with SHD. If VAs originate away from the myocardial scar, they are considered idiopathic. Therefore, an imaging study such as echocardiography, nuclear test, or cardiac magnetic resonance imaging (cMRI) should be performed to locate the site of the scar in patients with SHD. Frequent IVAs can cause tachycardia-induced cardiomyopathy. When evidence of myocardial scar is excluded by a nuclear test or cMRI despite a reduced LV function, tachycardia-induced cardiomyopathy is likely to be present. A definite diagnosis of tachycardia-induced cardiomyopathy can be made when the LV function recovers after the IVAs are well treated by medication or catheter ablation.

3.2. Electrocardiogram

IVAs usually originate from specific anatomical structures and exhibit characteristic electrocardiograms (ECGs) based on their anatomical background. In general, the first clue in 12-lead surface electrocardiograms for predicting a site of an IVA origin is a bundle branch block pattern in lead V1. A right bundle branch block (RBBB) pattern clearly suggests an origin in the LV, whereas a left bundle branch block (LBBB) pattern suggests an origin in the RV or the interventricular septum. Second, an inferior axis (dominant R waves in leads II, III, and aVF) suggests an origin in the superior aspect of the ventricle, whereas a superior axis suggests an origin in the inferior aspect. A negative QRS polarity in lead I suggests an origin in the LV free wall [2, 9], and a QS pattern in lead V6 suggests an origin near the apex

(**Figures 4**, **8**, and **9**) [2, 21]. An R/S wave amplitude ratio of >1 in lead V6 suggests an origin in the base (ventricular outflow tract or annuli), whereas an R/S wave amplitude ratio of <1 suggests an origin in the middle of the ventricle (papillary muscles or left fascicles) (**Figures 4**, **8**, and **9**) [2, 21]. Twelve-lead ECGs are very helpful for predicting the likely epicardial VT origins (**Figures 10** and **11**). Because in human hearts, the Purkinje network that can quickly facilitate ventricular activation throughout the ventricles is located only in the subendocardium, ventricular activation from the epicardial origin requires more time to reach the Purkinje network, resulting in a slow onset of the QRS during epicardial VTs. Based on this mechanism, several parameters predicting epicardial VT origins have been proposed: a "pseudo-delta" wave duration of >34 ms, a QRS duration of >200 ms, a delayed intrinsicoid deflection of >85 ms, an RS complex duration of >121 ms, and a maximum deflection index (MDI) (calculated by dividing the shortest time from the QRS onset to the maximum deflection in any of the precordial leads by the total QRS duration) of >0.54 (**Figure 10**) [33, 34]. When ventricular activation propagates from an epicardial origin at the LV free wall or ventricular posterior wall, the total activation vector should go from a lateral toward medial or from an inferior toward superior direction, resulting in a QS pattern in lead I or lead aVF (**Figure 11**) [32]. On the other hand, when ventricular activation propagates from an

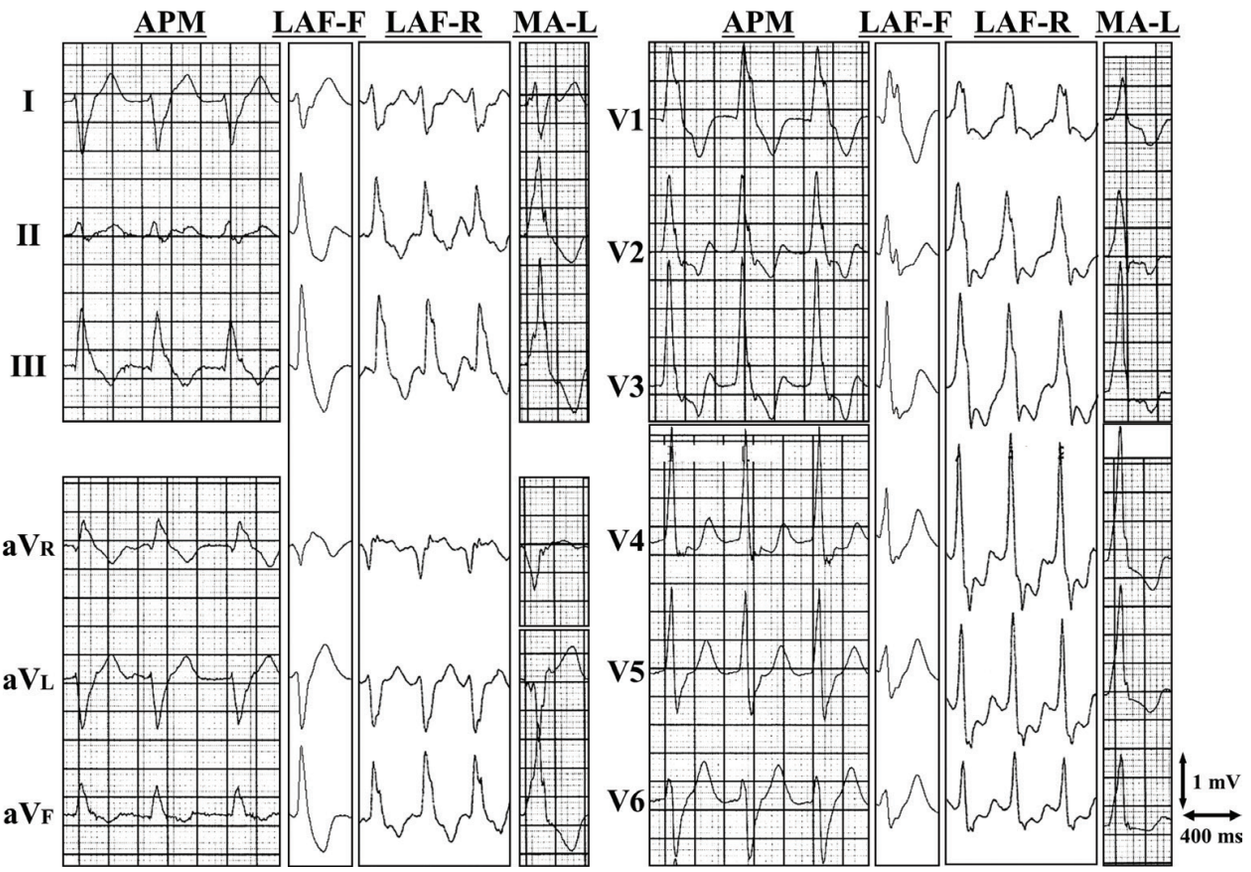


Figure 8. Representative 12-lead electrocardiograms of the QRS complexes during ventricular arrhythmias originating from the anterolateral region in the LV. APM, anterolateral papillary muscle; L, lateral portion; LAF, the left anterior fascicle; MA, mitral annulus; X-F, R, VAs with a focal or a macroreentrant mechanism. This figure was reproduced from Ref. [21] with permission.

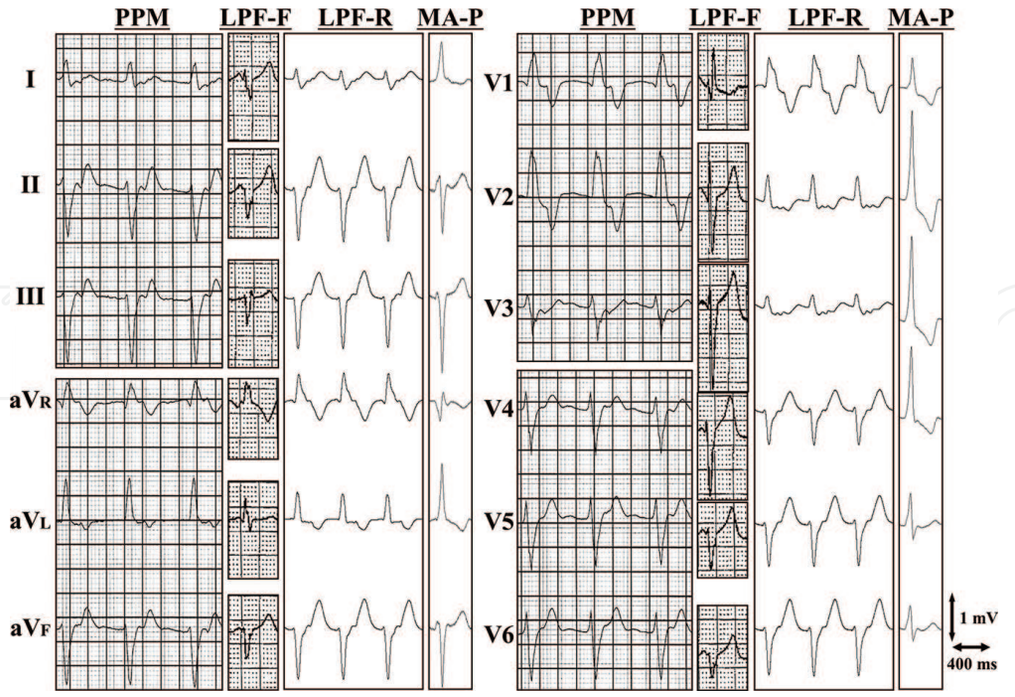


Figure 9. Representative 12-lead electrocardiograms of the QRS complexes during ventricular arrhythmias originating from the posteroseptal region in the LV. LPF, the left posterior fascicle; MA, mitral annulus; P, posterior portion; PPM, posteromedial papillary muscle; X-F, R, VAs with a focal or a macroreentrant mechanism. This figure was reproduced from Ref. [21] with permission.

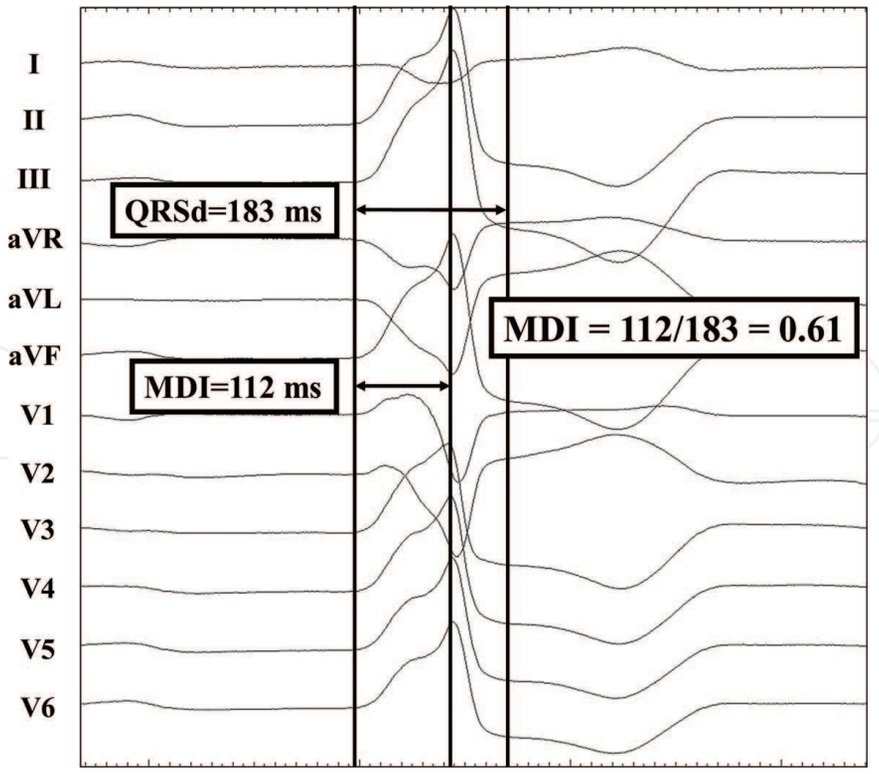


Figure 10. Twelve-lead electrocardiograms exhibiting a ventricular arrhythmia originating from the LV summit and the measurement of the maximal deflection index (MDI). This figure was cited from Ref. [3] with permission.

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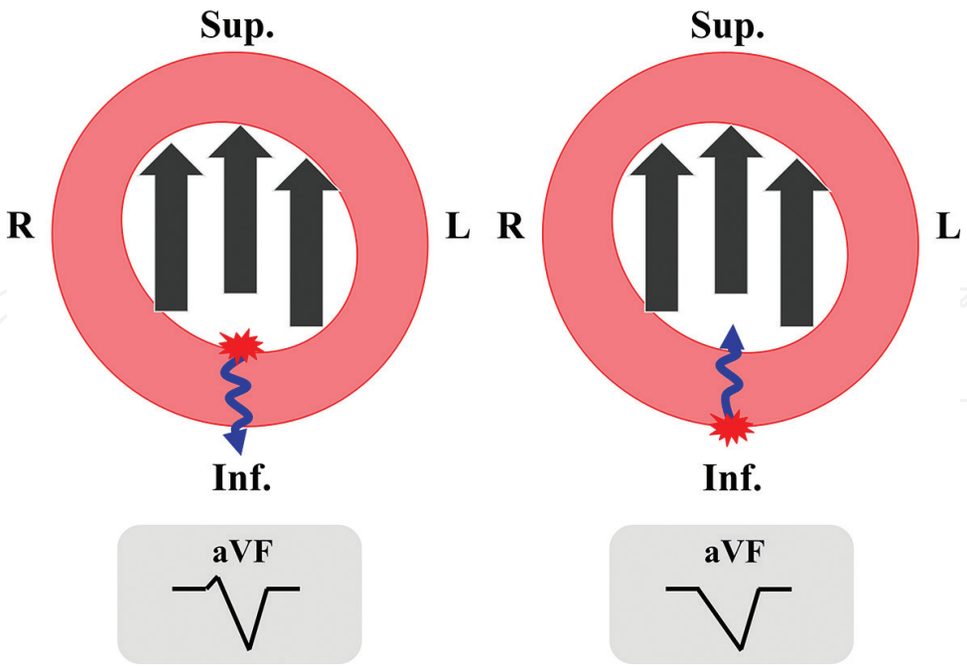


Figure 11. Schema showing the mechanism to explain the difference in the QRS morphology in lead aVF during ventricular tachycardias with endocardial (left) and epicardial (right) foci. Inf, inferior; L, left; R, right; Sup, superior. This figure was cited from Ref. [32] with permission.

endocardial origin on the LV free wall or ventricular posterior wall, a part of the activation vector should go toward the lateral or the inferior direction, which reflects the activation conducting through the wall of the ventricular muscle toward the epicardium, resulting in the presence of an initial R wave in lead I or lead aVF (**Figure 11**). Therefore, a QS pattern in lead I or aVF suggests an epicardial origin in the LV free wall [10] or the ventricular posterior wall, respectively (**Figure 11**). All these ECG features are more accurate without SHD than with it, because without any scar tissue associated with SHD, the ventricular activation propagates away from VA origins through normal ventricular myocardium in a predictable manner.

IVAs originating from the RVOT and LVOT exhibit similar ECG characteristics because anatomically, the RVOT and LVOT are located next to each other (**Figure 3**). The ECGs of idiopathic outflow tract VAs are characterized by positive R waves in all inferior leads and deep S waves in both leads aVR and aVL (almost QS pattern) (**Figures 12–14**). An RBBB QRS morphology clearly suggests a VA origin on the left side. However, when idiopathic outflow tract VAs exhibit an LBBB QRS morphology, it is often difficult to differentiate RVOT VAs from LVOT VAs. Because anatomically, the LVOT is located posterior to the RVOT (**Figure 3**), LVOT VAs exhibit taller and wider R waves in leads V1 and V2 than RVOT VAs. Therefore, the precordial transition is helpful for differentiating RVOT VAs from LVOT VAs. When the precordial transition is later than lead V4, the VAs are very likely to originate from the RVOT, and when the precordial transition is earlier than lead V2, the VAs are very likely to originate from the LVOT. However, when the precordial transition is in lead V3, it is most difficult to differentiate RVOT VAs from LVOT VAs. Although multiple ECG algorithms to differentiate RVOT VAs from LVOT VAs have been proposed, two ECG algorithms may be recommended, the magnitude and width of the R wave or QRS complex in leads V1 and V2

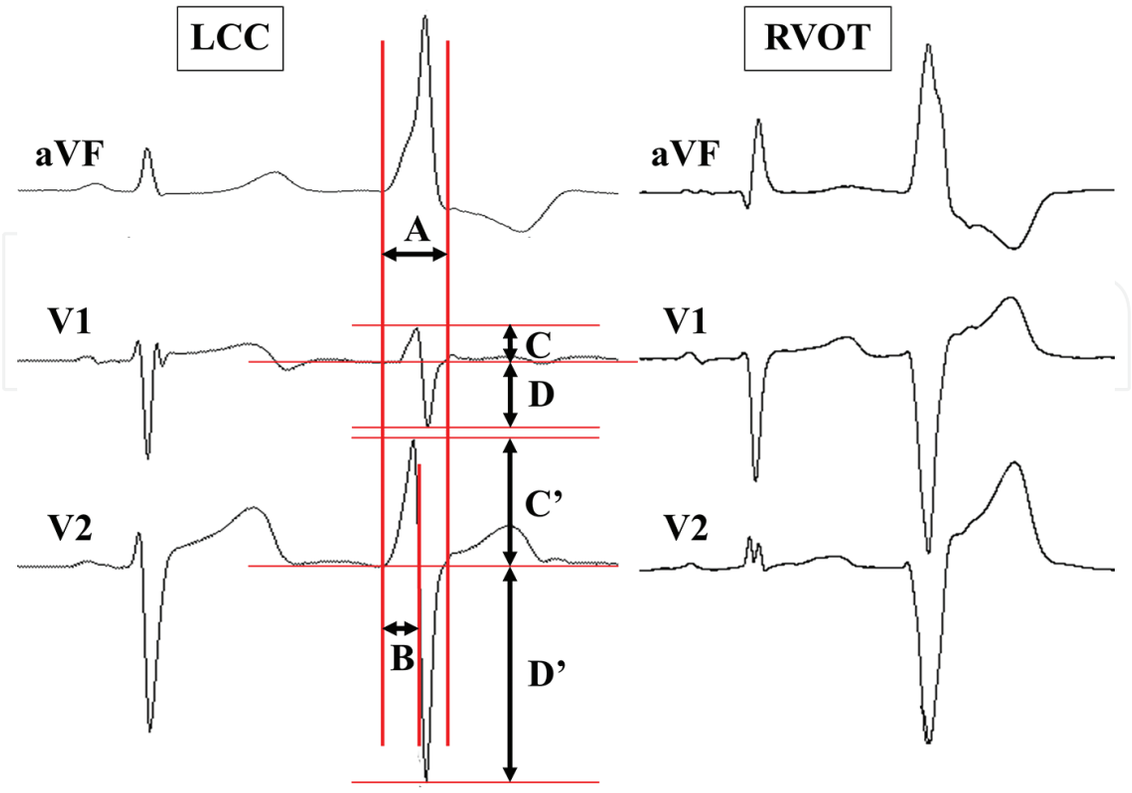


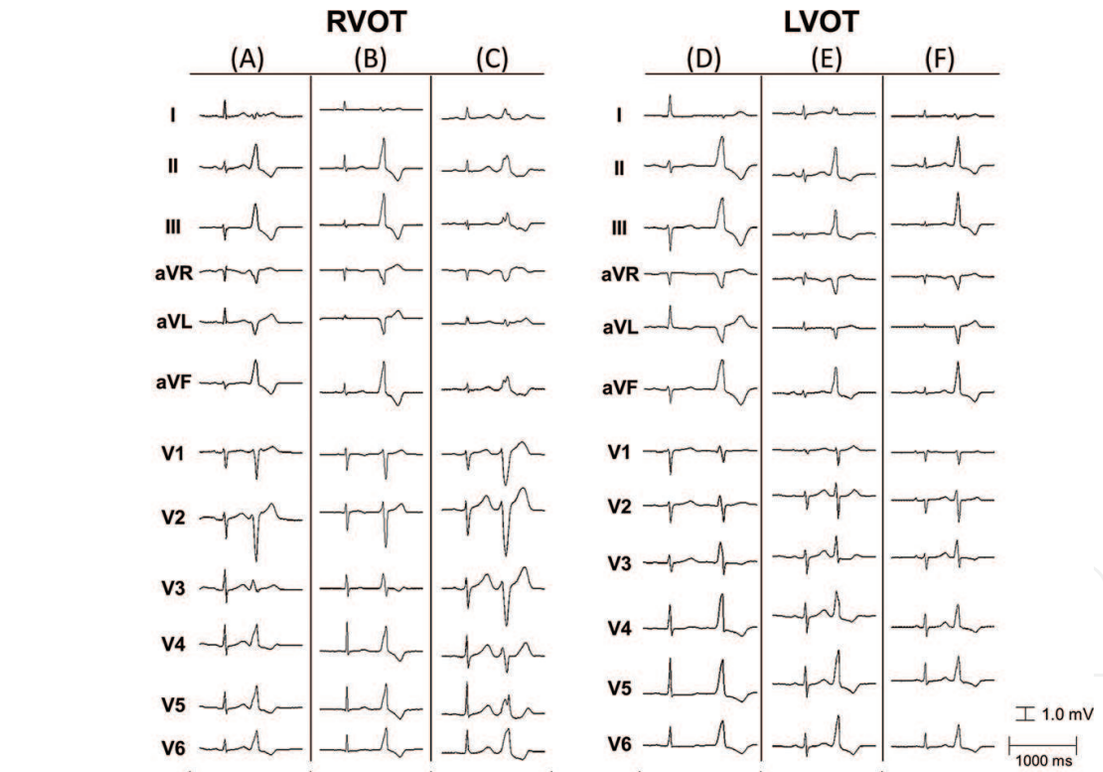
Figure 12. Examples of an electrocardiographic analysis of ventricular arrhythmias. The first beats are sinus and the second beats are ventricular arrhythmias originating from the left coronary cusp (LCC) and the right ventricular outflow tract (RVOT). **A** indicates the total QRS duration, **B** the longer R-wave duration in lead V1 or V2, determined in lead V2 from the QRS onset to the R-wave intersection point where the R-wave crosses the isoelectric line, **C** the R-wave amplitude, measured from the peak to the isoelectric line, and **D** the S-wave amplitude measured from the QRS nadir to the isoelectric line. The R/S wave amplitude ratio in lead V2 (C′/D′) is greater than that in lead V1 (C/D), and C′/D′ is determined as the R/S wave amplitude index. The R/S amplitude index is less than 0.3 and the R-wave duration index (B/A) less than 0.5 during RVOT VAs, whereas they are not during LCC VAs. This figure was cited from Ref. [6] with permission.

(R/S wave amplitude and duration indexes) [8] (**Figure 12**) and V2S/V3R amplitude ratio [35] (**Figure 13**), because they can simply and accurately perform a diagnosis by an ECG of VA only. The R/S wave amplitude in leads V1 and V2 is measured as the amplitude of the QRS complex peak or nadir to the isoelectric line. The R/S wave amplitude index, calculated from the percentage of the R/S wave amplitude ratio in lead V1 or V2 (whichever is greater), is considered more useful than the R/S wave amplitude ratio alone in lead V1 or V2. The R-wave duration index is calculated by dividing the longer R-wave duration in lead V1 or V2 by the QRS complex duration. An R/S amplitude index of <0.3 and an R-wave duration index of <0.5 may strongly suggest a VA origin on the right side (**Figure 13**) [8]. The V2S/V3R amplitude ratio is calculated by dividing the amplitude of the S wave in lead V2 by that of R wave in lead V3. A V2S/V3R amplitude ratio of ≤1.5 can predict LVOT VA origins and that of >1.5 RVOT VA origins (**Figure 13**) [35]. This ECG algorithm is useful even when the precordial transition is observed in lead V3 and has been proven to be the most accurate among the previous ECG algorithms to differentiate RVOT VA origins from LVOT VA origins.

Although the three ASCs are located next to each other, IVAs that can be ablated within each ASC may be differentiated by ECGs (**Figure 14**) [7]. IVAs that can be ablated within the RCC

and at the L-RCC rarely exhibit an RBBB pattern, and IVAs that can be ablated within the NCC always exhibit an LBBB pattern. The R-wave amplitude ratio in leads III–II (III/II ratio) is useful for differentiating LCC VAs from RCC VAs. When the III/II ratio is >0.9, VAs are more likely to be ablated within the LCC. A qrS pattern in the right precordial leads may be highly specific for an L-RCC VA origin (**Figure 14**) [13]. The ECG characteristics of NCC VAs are similar to those of RCC VAs (**Figure 14**) [14]. However, an S wave in lead III is present during NCC VAs although it is not during RCC VAs. When the III/II ratio is <0.65, VAs are more likely to be ablated from within the NCC.

All MA VAs exhibit an RBBB pattern and monophasic R or Rs in leads V2–V6 (**Figure 15**) [9, 10]. Because the origins of all MA VAs are located in the posterior portion of the LV, which is distant from the precordial electrodes, the activation from the MA VA origins propagates toward these electrodes, resulting in an early precordial transition and a concordant positive QRS pattern in leads V2–V4 during MA VAs. The ECG characteristics are very helpful for predicting sites of MA VA origins [9, 10]. The polarity of the QRS complex in the inferior and lateral leads (I and aVL) is positive and negative in anterolateral MA VAs, while it is negative



	RVOT			LVOT		
	(A)	(B)	(C)	(D)	(E)	(F)
S amp in V2 (mV)	3.10	2.59	3.30	1.24	1.67	1.49
R amp in V3 (mV)	0.76	1.06	0.54	1.90	1.52	1.31
V2S/V3R index	4.08	2.44	6.11	0.65	1.10	1.14

Figure 13. Representative 12-lead electrocardiograms of VAs originating from the ventricular outflow tract. The first beat is a sinus beat and the second is a premature ventricular contraction in each panel (A–F). The S-wave amplitude in lead V2, R-wave amplitude in lead V3, and V2S/V3R index are listed below each panel. All right ventricular outflow tract (RVOT) PVCs exhibited a V2S/V3R index of >1.5, while all left ventricular outflow tract (LVOT) PVCs exhibited a V2S/V3R index of ≤1.5. The PVCs were successfully ablated in the RVOT septum (A and B), RVOT free wall (C), left coronary cusp (D), right coronary cusp (E), and aorto-mitral continuity (F). The other abbreviation is as in the previous figure. This figure was cited from Ref. [35] with permission.

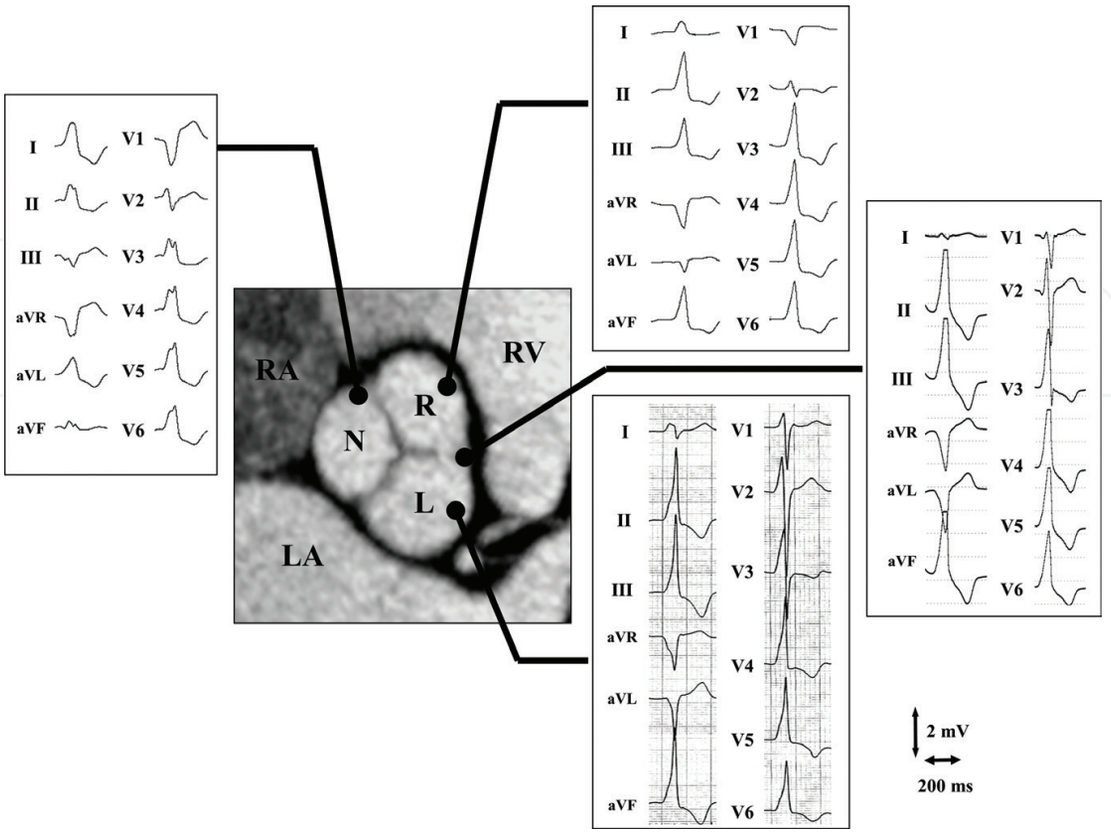


Figure 14. Two-dimensional CT images and representative 12-lead electrocardiograms of ventricular arrhythmias originating from the aortic root. L, left coronary cusp; N, noncoronary cusp; R, right coronary cusp. The other abbreviations are as in the previous figures. This figure was cited from Ref. [7] with permission.

and positive in posterior and posterolateral MA VAs, respectively. MA VAs originating from the free wall of the MA are characterized by a longer QRS duration sometimes with pseudo-delta waves and notching in the late phase of the R or Q wave in the inferior leads, which may result from phased excitation from the LV free wall to the RV (**Figure 15**). Posterior MA VAs exhibit a dominant R wave in lead V1, whereas posteroseptal MA VAs exhibit a negative QRS component in lead V1 (qR, qr, rs, rS, or QS).

All TA VAs exhibit an LBBB QRS morphology and positive QRS polarity in leads I, V5, and V6 (**Figure 16**) [17] because the TA VA origins are located on the right anterior side of the heart, and the activation propagating from TA VA origins toward the apex generates a positive QRS polarity in leads V5 and V6. The R wave in lead I is usually taller during TA VAs than during RVOT VAs because the TA is located more rightward and inferior to the RVOT. For the same reason, a positive QRS polarity in all of the inferior leads is rare in TA VAs but common in all RVOT VAs. During TA VAs, a QS or an rS pattern in lead aVL is rare, and the QRS polarity in lead aVL is positive in almost all TA VAs, which is not the case for RVOT VAs. Among all TA VAs, the QRS duration and Q wave amplitude in each of the leads V1–V3 are greater in TA VAs originating from the free wall of the TA than in those from the septal wall of the TA [17]. Septal TA VAs exhibit an early precordial transition (lead V3), a narrower QRS duration, and QS in lead V1 with the absence of notching in the inferior leads while the free wall TA VAs are associated with a late precordial transition (>lead V3), a wider QRS duration, the absence of Q

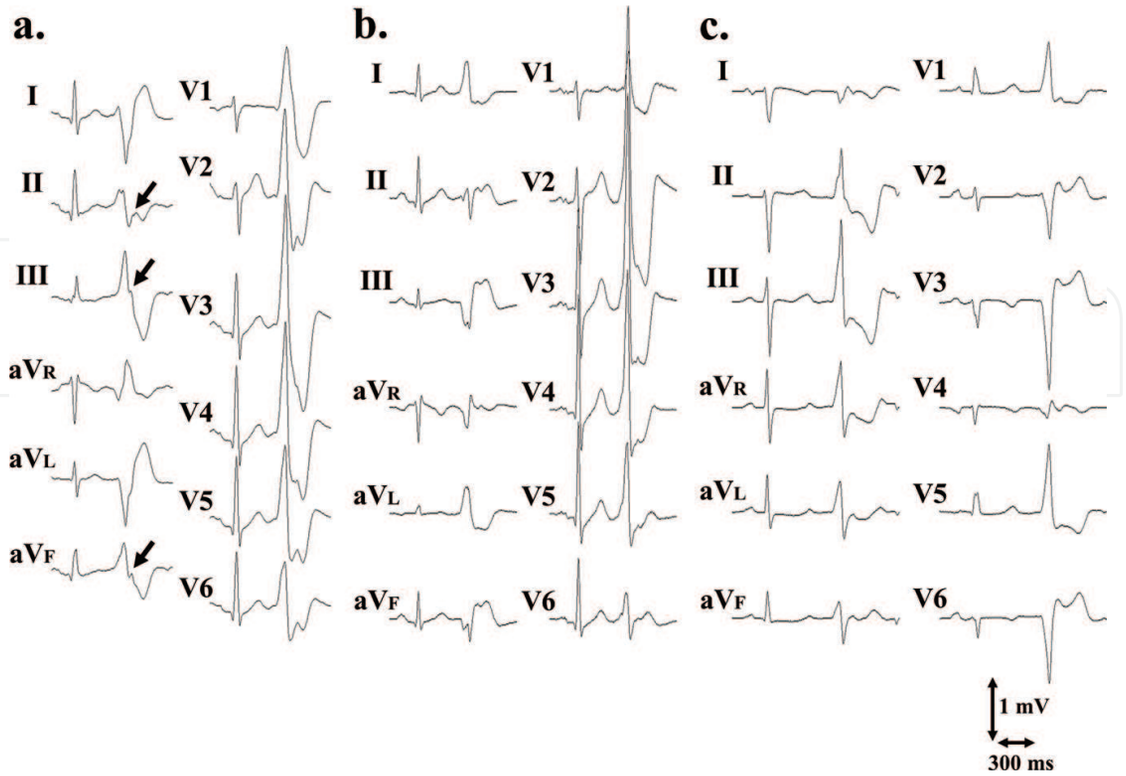


Figure 15. Representative 12-lead electrocardiograms of the premature ventricular contractions originating from the anterolateral (a), posterior (b), and posteroseptal (c) aspects of the mitral annulus. The arrows indicate "notching" of the late phase of the QRS complex in the inferior leads. This figure was cited from Ref. [10] with permission.

waves in lead V1, and the presence of notching in the inferior leads (the timing of the second peak of the notched QRS complex in the inferior leads corresponds precisely with the LV free wall activation) (**Figure 16**). A negative QRS polarity in the inferior leads predicts VA origins in the posterior aspect of the TA, and otherwise, VA origins in the mid- to anterior aspects of the TA are suggested.

IVAs originating from the anterolateral and posteromedial PAMs in the LV exhibit RBBB and right inferior and left or right superior axis QRS morphologies, respectively (**Figures 8** and **9**) [18–21]. IVAs originating from the posterior or anterior RV PAMs more often exhibit a superior axis with a late precordial transition (>lead V4) as compared with septal RV PAM VAs, which more often exhibit an inferior axis with an earlier precordial transition (≤lead V4) [22].

Because of the close anatomical relationship, it is important to distinguish PAM VAs from MA VAs and LV fascicular VAs by ECGs (**Figures 8** and **9**) [21]. The ECG features such as an rS in lead I, an rS in lead aVR (for only the LV anterolateral region), a qR in lead aVL, a Q in lead V1, an S wave amplitude ratio in leads III to II <1.5, and an R/S ratio of ≤1 in lead V6 (the last two parameters are for only the LV posteroseptal region) can accurately distinguish MA VAs from PAM and LV fascicular VAs [21]. However, the ECG features are very similar for PAM and LV fascicular VAs, and an R/S ratio of ≤1 in lead V6 in the LV anterolateral region and a QRS duration of >160 ms, and qR or R waves in lead V1 (as compared with an rsR′ for fascicular VTs) in the LV posteroseptal region may be the only reliable predictors for differentiating PAM VAs from LV fascicular VAs [21].

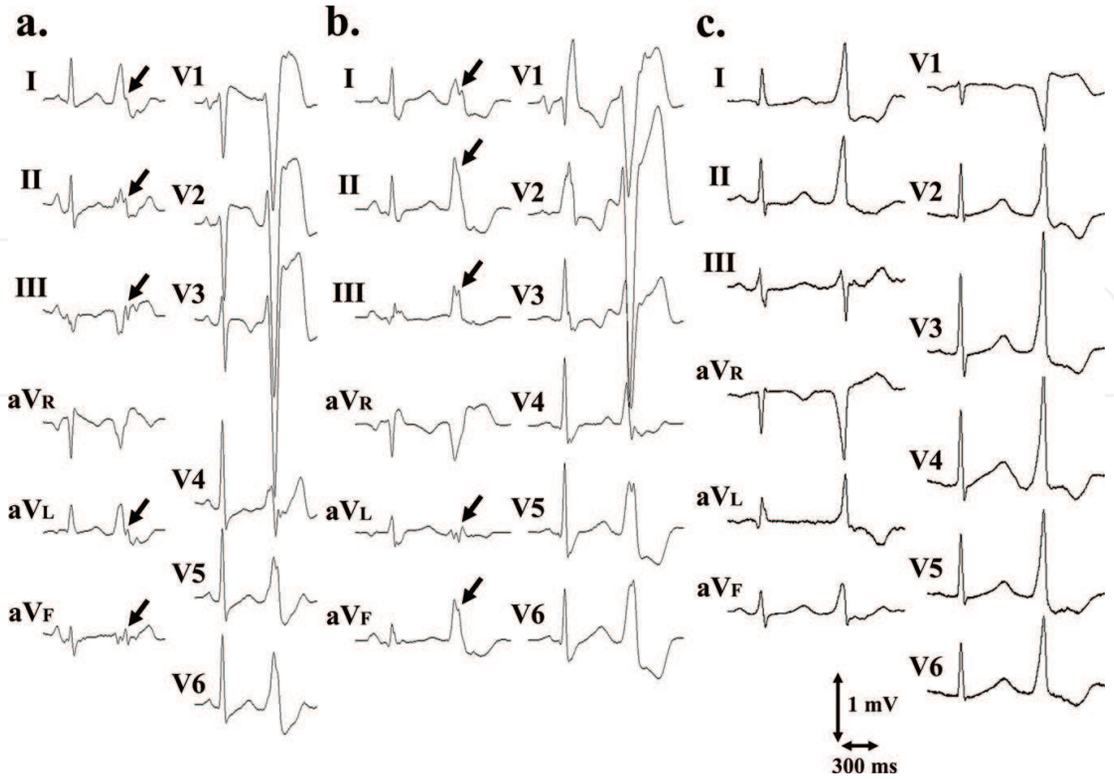


Figure 16. Representative 12-lead electrocardiograms of the premature ventricular contractions originating from the posterolateral (a), anterior (b), and anteroseptal (c) aspects of the tricuspid annulus. The arrows indicate "notching" of the late phase of the QRS complex in the limb leads. This figure was cited from Ref. [10] with permission.

IVAs arising from the MB exhibit a distinctive ECG morphology, LBBB and left superior axis QRS morphology, a sharp downstroke of the QRS in the precordial leads, and a relatively narrow QRS duration (**Figure 4**) [23]. MB VAs not only have a late precordial transition pattern, typically after lead V4, but also the transition is always later than that of the sinus QRS. Among the idiopathic RV VAs, a late precordial transition and a superiorly directed nature are helpful for distinguishing MB VAs from VAs originating from the RV base or septum [23]. The ECG characteristics of the IVAs originating from the infundibular muscles are similar to those of IVAs originating from the RVOT and the anterior to anteroseptal aspect of the TA [24, 25]. However, the precordial transition is relatively early, and a slow onset of the QRS complex is often observed.

IVAs arising from the crux of the heart exhibit a left superior axis QRS morphology with deeply negative deltoid waves (QS pattern) in the inferior leads and an early precordial transition (a prominent R wave in lead V2), which may be associated with a polarity reversal between leads V1 and V2 (**Figure 6**) [30]. It is noted that crux VAs often exhibit a QS or a large S wave in lead V6 although they arise from the LV base. This is likely because the activation from the crux VA origins first conducts to the ventricular apex where it enters the Purkinje system and then propagates throughout the ventricles. The common ECG characteristics of LV summit VAs are a right inferior axis QRS morphology, a wider QRS, and a larger MDI than the other idiopathic LVOT VAs [31]. The MDI [34] of these epicardial IVAs is usually >0.55. The AMC and LV summit face each other with the superior end of the LV muscle between them, which is attached to the LCC. Because of the anatomical proximity, the presence of

preferential conduction, and the presence of intramural VA origins, it is challenging to predict where LVOT VAs can be ablated among those three sites by an ECG algorithm [28, 29].

4. Treatment of IVAs

Treatment of IVAs should be tailored according to the presentation type of VAs, PVCs, or VTs, and the patient characteristics (**Figure 17**) [4, 5]. When SHD is absent, the most common indication for treating PVCs remains the presence of symptoms. The severity of the symptoms from PVCs is not closely related to the frequency of PVCs. Even when the PVCs are infrequent, some patients are very symptomatic. When PVCs are not frequent, the physician has to explain and reassure that there is a benign nature of idiopathic PVCs. It is a common experience that symptoms from PVCs can improve without any treatment in most patients with infrequent PVCs. Exercise stress testing should be considered to determine whether PVCs are potentiated or suppressed by exercise, to assess whether longer duration VAs are provoked especially when symptoms are associated with exercise. PVCs that worsen with exercise should prompt further investigation as these patients are more likely to require treatment. Frequent asymptomatic PVCs may have to be treated if PVC-induced cardiomyopathy

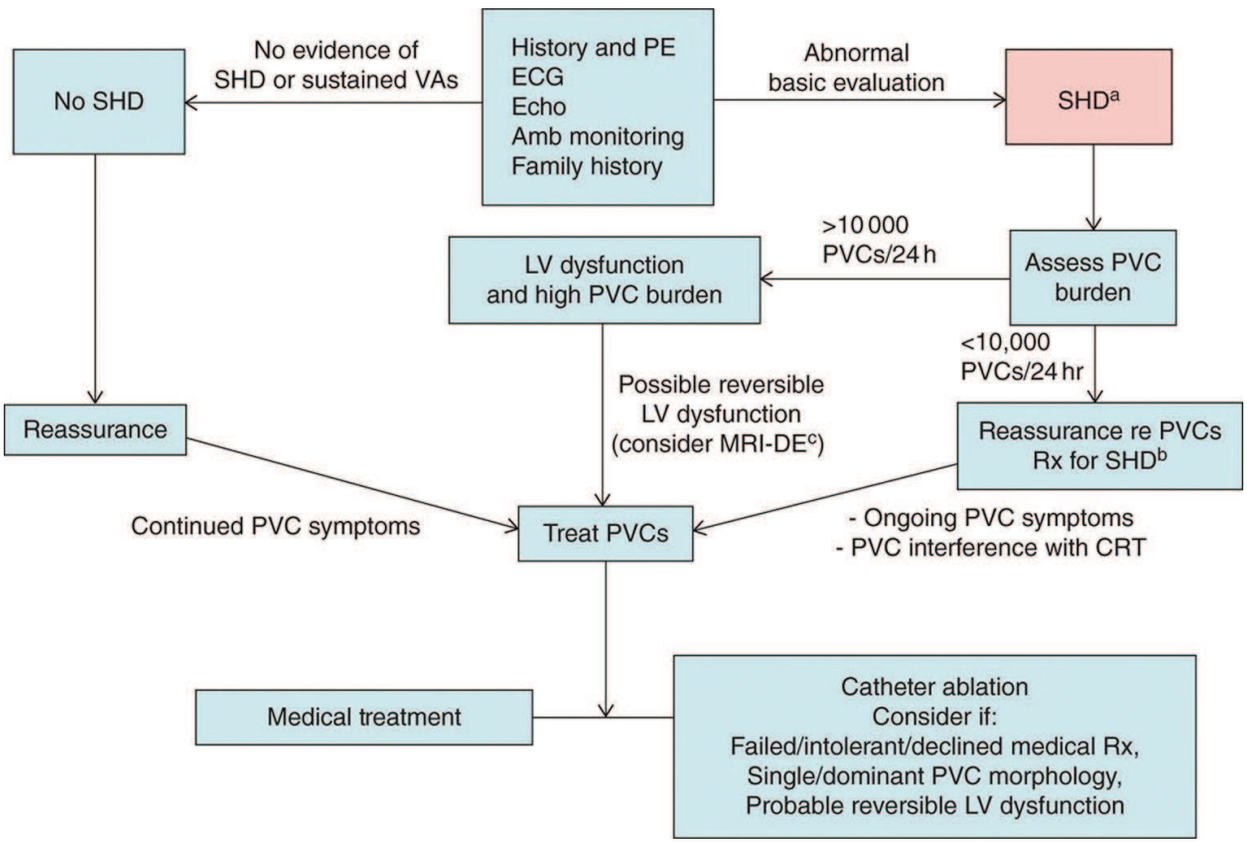


Figure 17. Schema exhibiting the management of premature ventricular contractions (PVCs). (a) Absence of a high-scar burden suggests reversibility; (b) medical therapy + implantable cardioverter-defibrillator. CRT, cardiac resynchronization therapy; LV, left ventricular; MRI-DE, magnetic resonance imaging with delayed enhancement; PE, physical examination; Rx, therapy; SHD, structural heart disease; VAs, ventricular arrhythmias. This figure was cited from Ref. [5] with permission.

is present. When a PVC burden is greater than 10% (approximately 10,000 PVCs/24 h), the risk of PVC-induced cardiomyopathy is significant. Therefore, such high-burdened PVCs have to be treated when they are symptomatic. When they are asymptomatic, a close follow-up with repeat echocardiography and Holter monitoring should be considered to detect any occurrence of PVC-induced cardiomyopathy. In patients with fewer PVCs, further investigation is only necessary should the symptoms increase. For patients without SHD and mild symptoms, education of the benign nature of this arrhythmia and reassurance should be considered as the first step in the treatment of patients with PVCs. For patients whose symptoms are not effectively managed in this manner, beta-blockers or non-dihydropyridine calcium antagonists may be attempted although the efficacy of these agents is quite limited with only 10–15% of patients achieving a 90% PVC suppression, similar to placebo [4, 5]. It should also be recognized that these agents may themselves produce significant side effects rather than relieve the PVC symptoms. Membrane-active anti-arrhythmic drugs (AADs) are more effective for suppressing PVCs and can be attempted when beta-blockers or non-dihydropyridine calcium antagonists are not effective. Because these agents may increase the risk of mortality in patients with significant SHD, perhaps with the exception of amiodarone, caution is advised before using them for PVC suppression.

When idiopathic PVCs are refractory to medication or patients cannot tolerate medication, catheter ablation can be a next option for their treatment. Randomized trials of PVC suppression with catheter ablation have not been performed. However, multiple studies have revealed that catheter ablation is highly successful with PVC elimination in 74–100% of highly symptomatic patients with a very high PVC burden [4, 5]. Procedural success may be dependent on the site of the VA origin with a lower efficacy reported for IVAs with epicardial foci and anatomical challenges than for other IVAs [1–5]. Although complete PVC elimination is the goal of ablation, partial success with a significant reduction in the PVC burden may still be associated with significant improvement in the symptoms as well as LV systolic function. Catheter ablation of IVAs may be less successful when multiple morphologies of PVCs present or the clinical PVC morphology cannot be induced at the time of the procedure [1–5]. The published complication rates of catheter ablation for PVC suppression are generally low (<1%) [1–5]. According to the current recommendations of the experts' consensus, catheter ablation of PVCs may be considered for highly selected patients who remain very symptomatic despite conservative treatment or for those with high PVC burdens associated with a decline in the LV systolic function [4, 5].

Idiopathic VTs are basically monomorphic and hemodynamically stable. When SHD is absent, sustained idiopathic VTs are generally associated with an excellent prognosis [1, 4, 5]. Idiopathic VTs rarely can have a malignant clinical course, usually with a very rapid rate or a short initiating coupling interval [1, 4, 5]. Idiopathic non-sustained VTs (NSVTs) usually present with frequent PVCs with the same QRS morphology, and most of them originate from the RVOT or LVOT. These arrhythmias only require treatment if they are symptomatic, incessant, or produce LV dysfunction. The treatment of these VTs is either medical with beta-blockers, non-hydropyridine calcium blockers, or class IC drugs, or catheter ablation with a high success rate and low risk of complications [1, 4, 5]. Non-sustained and sustained VTs with a focal mechanism likely based on abnormal automaticity may also occur from the papillary

muscles and respond to beta-blockers or catheter ablation with a relatively low success rate [4, 5, 18–21]. Reentrant LV fascicular VTs usually present as a sustained form and can be acutely treated with intravenous verapamil or mexiletine. Oral therapy with these medicines can be used to prevent recurrence of those VTs, although the recurrence risk may be relatively high [4, 5, 26, 27]. Catheter ablation can be recommended when idiopathic VTs are highly symptomatic and drug-refractory, especially if they are exercise-induced [1, 4, 5].

Catheter ablation of IVAs is usually safe and highly successful, but sometimes can be challenging because of the anatomical obstacles such as close proximity to the coronary arteries and AV conduction system, epicardial fat pads, intramural and epicardial origins, and thick muscle bands. Understanding the relevant anatomy is helpful for achieving a safe and successful catheter ablation of IVAs.

5. Conclusions

The sites of IVA origins have been increasingly recognized for the past two decades. IVAs usually originate from specific anatomical structures, commonly endocardial but sometimes epicardial, and exhibit characteristic ECGs based on their anatomical background. IVAs are basically benign, but they require medical treatment or catheter ablation when IVAs are symptomatic, incessant, or produce LV dysfunction.

Conflict of interest

The author declares no conflicts of interest.

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References

[1] Stevenson WG, Soejima K. Catheter ablation for ventricular tachycardia. Circulation. 2007;**115**:2750-2760

[2] Yamada T, Kay GN. Optimal ablation strategies for different types of ventricular tachycardias. Nature Reviews. Cardiology. 2012;**9**:512-525

[3] Yamada T. Idiopathic ventricular arrhythmias: Relevance to the anatomy, diagnosis and treatment. Journal of Cardiology. 2016;**68**:463-471

[4] Pedersen CT, Kay GN, Kalman J, Borggrefe M, Della-Bella P, Dickfeld T, Dorian P, Huikuri H, Kim YH, Knight B, Marchlinski F, Ross D, Sacher F, Sapp J, Shivkumar K, Soejima K, Tada H, Alexander ME, Triedman JK, Yamada T, Kirchhof P, Document Reviewers, Lip GY, Kuck KH, Mont L, Haines D, Indik J, Dimarco J, Exner D, Iesaka Y, Savelieva I. EHRA/HRS/APHRS expert consensus on ventricular arrhythmias. Europace. 2014;**16**:1257-1283

[5] Pedersen CT, Kay GN, Kalman J, Borggrefe M, Della-Bella P, Dickfeld T, Dorian P, Huikuri H, Kim YH, Knight B, Marchlinski F, Ross D, Sacher F, Sapp J, Shivkumar K, Soejima K, Tada H, Alexander ME, Triedman JK, Yamada T, Kirchhof P, Lip GY, Kuck KH, Mont L, Haines D, Indik J, Dimarco J, Exner D, Iesaka Y, Savelieva I, EP-Europace, UK. EHRA/HRS/APHRS expert consensus on ventricular arrhythmias. Heart Rhythm. 2014;**11**:e166-e196

[6] Yamada T, Kay GN. How to diagnose and ablate ventricular tachycardia from the outflow tract and aortic cusps. In: Al-Ahmad A, Callans DJ, Hsia HH, Natale A, Oseroff O, Wang PJ, editors. Hands-On Ablation: The Experts' Approach. 2nd ed. Minneapolis, MN: Cardiotext Publishing; 2017. pp. 292-301

[7] Yamada T, McElderry HT, Doppalapudi H, Murakami Y, Yoshida Y, Yoshida N, Okada T, Tsuboi N, Inden Y, Murohara T, Epstein AE, Plumb VJ, Singh SP, Kay GN. Idiopathic ventricular arrhythmias originating from the aortic root: Prevalence, electrocardiographic and electrophysiological characteristics, and results of the radiofrequency catheter ablation. Journal of the American College of Cardiology. 2008;**52**:139-147

[8] Ouyang F, Fotuhi P, Ho SY, Hebe J, Volkmer M, Goya M, Burns M, Antz M, Ernst S, Cappato R, Kuck KH. Repetitive monomorphic ventricular tachycardia originating from the aortic sinus cusp: Electrocardiographic characterization for guiding catheter ablation. Journal of the American College of Cardiology. 2002;**39**:500-508

[9] Tada H, Ito S, Naito S, Kurosaki K, Kubota S, Sugiyasu A, Tsuchiya T, Miyaji K, Yamada M, Kutsumi Y, Oshima S, Nogami A, Taniguchi K. Idiopathic ventricular arrhythmia arising from the mitral annulus: A distinct subgroup of idiopathic ventricular arrhythmias. Journal of the American College of Cardiology. 2005;**45**:877-886

[10] Yamada T. Idiopathic ventricular tachycardia from mitral annulus, papillary muscles and other sites. In: Bhargava K, Asirvatham SJ. Practical Cardiac Electrophysiology. Jaypee Brothers Medical Publishers Pvt. Ltd.; 2016. pp. 543-66

[11] McAlpine WA. Heart and Coronary Arteries. New York: Springer-Verlag; 1975

[12] Yamada T, Litovsky SH, Kay GN. The left ventricular ostium: An anatomic concept relevant to idiopathic ventricular arrhythmias. Circulation. Arrhythmia and Electrophysiology. 2008;**1**:396-404

[13] Yamada T, Yoshida N, Murakami Y, Okada T, Muto M, Murohara T, McElderry HT, Kay GN. Electrocardiographic characteristics of ventricular arrhythmias originating from the

junction of the left and right coronary sinuses of Valsalva in the aorta: The activation pattern as a rationale for the electrocardiographic characteristics. Heart Rhythm. 2008;**5**:184-192

[14] Yamada T, Lau YR, Litovsky SH, Thomas McElderry H, Doppalapudi H, Osorio J, Plumb VJ, Neal Kay G. Prevalence and clinical, electrocardiographic, and electrophysiologic characteristics of ventricular arrhythmias originating from the noncoronary sinus of Valsalva. Heart Rhythm. 2013;**10**:1605-1612

[15] Yamada T, McElderry HT, Doppalapudi H, Kay GN. Catheter ablation of ventricular arrhythmias originating in the vicinity of the His bundle: Significance of mapping the aortic sinus cusp. Heart Rhythm. 2008;**5**:37-42

[16] Sekiguchi Y, Aonuma K, Takahashi A, Yamauchi Y, Hachiya H, Yokoyama Y, Iesaka Y, Isobe M. Electrocardiographic and electrophysiologic characteristics of ventricular tachycardia originating within the pulmonary artery. Journal of the American College of Cardiology. 2005;**45**:887-895

[17] Tada H, Tadokoro K, Ito S, Naito S, Hashimoto T, Kaseno K, Miyaji K, Sugiyasu A, Tsuchiya T, Kutsumi Y, Nogami A, Oshima S, Taniguchi K. Idiopathic ventricular arrhythmias originating from the tricuspid annulus: Prevalence, electrocardiographic characteristics, and results of radiofrequency catheter ablation. Heart Rhythm. 2007;**4**:7-16

[18] Doppalapudi H, Yamada T, McElderry HT, Plumb VJ, Epstein AE, Kay GN. Ventricular tachycardia originating from the posterior papillary muscle in the left ventricle: A distinct clinical syndrome. Circulation. Arrhythmia and Electrophysiology. 2008;**1**:23-29

[19] Yamada T, McElderry HT, Okada T, Murakami Y, Doppalapudi H, Yoshida N, Allred JD, Murohara T, Kay GN. Idiopathic focal ventricular arrhythmias originating from the anterior papillary muscle in the left ventricle. Journal of Cardiovascular Electrophysiology. 2009;**20**:866-872

[20] Yamada T, Doppalapudi H, McElderry HT, Okada T, Murakami Y, Inden Y, Yoshida Y, Yoshida N, Murohara T, Epstein AE, Plumb VJ, Litovsky SH, Kay GN. Electrocardiographic and electrophysiological characteristics in idiopathic ventricular arrhythmias originating from the papillary muscles in the left ventricle: Relevance for catheter ablation. Circulation. Arrhythmia and Electrophysiology. 2010;**3**:324-331

[21] Yamada T, Doppalapudi H, McElderry HT, Okada T, Murakami Y, Inden Y, Yoshida Y, Kaneko S, Yoshida N, Murohara T, Epstein AE, Plumb VJ, Kay GN. Idiopathic ventricular arrhythmias originating from the papillary muscles in the left ventricle: Prevalence, electrocardiographic and electrophysiological characteristics, and results of the radiofrequency catheter ablation. Journal of Cardiovascular Electrophysiology. 2010;**21**:62-69

[22] Crawford T, Mueller G, Good E, Jongnarangsin K, Chugh A, Pelosi F Jr, Ebinger M, Oral H, Morady F, Bogun F. Ventricular arrhythmias originating from papillary muscles in the right ventricle. Heart Rhythm. 2010;**7**:725-730

[23] Sadek MM, Benhayon D, Sureddi R, Chik W, Santangeli P, Supple GE, Hutchinson MD, Bala R, Carballeira L, Zado ES, Patel VV, Callans DJ, Marchlinski FE, Garcia FC. Idiopathic

ventricular arrhythmias originating from the moderator band: Electrocardiographic characteristics and treatment by catheter ablation. Heart Rhythm. 2015;**12**:67-75

[24] Yamada T, Yoshida N, Itoh T, Litovsky SH, Doppalapudi H, McElderry HT, Kay GN. Idiopathic ventricular arrhythmias originating from the parietal band: Electrocardiographic and electrophysiological characteristics and outcome of catheter ablation. Circulation. Arrhythmia and Electrophysiology. 2017;**10**:e005099

[25] Yamada T, Yoshida N, Litovsky SH, Itoh T, Doppalapudi H, Kay GN. Idiopathic ventricular arrhythmias originating from the infundibular muscles: Prevalence, electrocardiographic and electrophysiological characteristics and outcome of catheter ablation. Circulation: Arrhythmia and Electrophysiology. 2018;**11**:e005749

[26] Tsuchiya T, Okumura K, Honda T, Honda T, Iwasa A, Yasue H, Tabuchi T. Significance of late diastolic potential preceding Purkinje potential in verapamil-sensitive idiopathic left ventricular tachycardia. Circulation. 1999;**99**:2408-2413

[27] Nogami A, Naito S, Tada H, Taniguchi K, Okamoto Y, Nishimura S, Yamauchi Y, Aonuma K, Goya M, Iesaka Y, Hiroe M. Demonstration of diastolic and presystolic Purkinje potentials as critical potentials in a macroreentry circuit of verapamil-sensitive idiopathic left ventricular tachycardia. Journal of the American College of Cardiology. 2000;**36**:811-823

[28] Yamada T, Maddox WR, McElderry HT, Doppalapudi H, Plumb VJ, Kay GN. Radiofrequency catheter ablation of idiopathic ventricular arrhythmias originating from intramural foci in the left ventricular outflow tract: Efficacy of sequential versus simultaneous unipolar catheter ablation. Circulation. Arrhythmia and Electrophysiology. 2015;**8**:344-352

[29] Yamada T, Doppalapudi H, Maddox WR, McElderry HT, Plumb VJ, Kay GN. Prevalence and electrocardiographic and electrophysiological characteristics of idiopathic ventricular arrhythmias originating from intramural foci in the left ventricular outflow tract. Circulation: Arrhythmia and Electrophysiology. 2016;**9**:pii: e004079

[30] Doppalapudi H, Yamada T, Ramaswamy K, Ahn J, Kay GN. Idiopathic focal epicardial ventricular tachycardia originating from the crux of the heart. Heart Rhythm. 2009;**6**:44-50

[31] Yamada T, McElderry HT, Doppalapudi H, Okada T, Murakami Y, Yoshida Y, Yoshida N, Inden Y, Murohara T, Plumb VJ, Kay GN. Idiopathic ventricular arrhythmias originating from the left ventricular summit: Anatomic concepts relevant to ablation. Circulation. Arrhythmia and Electrophysiology. 2010;**3**:616-623.0

[32] Yamada T. Transthoracic epicardial catheter ablation: Indications, techniques, and complications. Circulation Journal. 2013;**77**:1672-1680

[33] Berruezo A, Mont L, Nava S, Chueca E, Bartholomay E, Brugada J. Electrocardiographic recognition of the epicardial origin of ventricular tachycardias. Circulation. 2004;**109**: 1842-1847

[34] Daniels DV, Lu YY, Morton JB, Santucci PA, Akar JG, Green A, Wilber DJ. Idiopathic epicardial left ventricular tachycardia originating remote from the sinus of Valsalva: Electrophysiological characteristics, catheter ablation, and identification from the 12-lead electrocardiogram. Circulation. 2006;**113**:1659-1666

[35] Yoshida N, Yamada T, McElderry HT, Inden Y, Shimano M, Murohara T, Kumar V, Doppalapudi H, Plumb VJ, Kay GN. A novel electrocardiographic criterion for differentiating a left from right ventricular outflow tract tachycardia origin: The V2S/V3R index. Journal of Cardiovascular Electrophysiology. 2014;**25**:747-753

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